Journal of the Formosan Medical Association (2017) xx, 1–9

Available online at www.sciencedirect.com

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journal homepage: www.jfma-online.com

ORIGINAL ARTICLE

Dual-phase ^{18}F-florbetapir positron emission tomography in patients with primary progressive aphasia, Alzheimer's disease, and healthy controls: A preliminary study

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Received 13 June 2016; received in revised form 16 February 2017; accepted 2 March 2017

KEYWORDS
^{18}F-florbetapir (AV-45/Amyvid amyloid imaging);
Alzheimer's disease;
logopenic variant;
primary progressive aphasia;
progressive nonfluent aphasia;

Abstract *Background:* To determine whether dual-phase ^{18}F-florbetapir positron emission tomography imaging with perfusion-like and amyloid deposition information can distinguish among primary progressive aphasia (PPA), Alzheimer's disease (AD), and healthy controls (HCs).
Methods: Patients diagnosed with PPA, including four semantic dementia (SD) and two progressive nonfluent aphasia (PNFA), as well as one logopenic variant (LV) of PPA, were studied. All PPA patients, and age-/sex-matched patients with probable AD ($n = 8$) and HCs ($n = 8$) were subjected to dual-phase ^{18}F-florbetapir imaging. Atlas-based quantitative volumes of interest (VOIs) analysis for six cortical areas and whole cerebellum was performed. The standardized uptake value ratios were calculated by normalizing the dual-phase-integrated activities of the six VOIs to whole cerebellum counts.

Conflicts of interest: All the authors declare no conflict of interest.

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http://dx.doi.org/10.1016/j.jfma.2017.03.003
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Please cite this article in press as: Kuo H-C, et al., Dual-phase ^{18}F-florbetapir positron emission tomography in patients with primary progressive aphasia, Alzheimer's disease, and healthy controls: A preliminary study, Journal of the Formosan Medical Association (2017), http://dx.doi.org/10.1016/j.jfma.2017.03.003

semantic dementia;
dementia;
clinical neurology;
diagnostic imaging;
radiology;
nuclear medicine and medical imaging

Results: Early phase ^{18}F-florbetapir image showed significantly lower global perfusion index in six PPA patients as compared with HCs. According to VOI analysis, the hypoperfusion lesions were identified in the frontal, anterior cingulate, parietal, precuneus, and temporal regions. Similar findings were confirmed by voxel-base image comparison. ^{18}F-florbetapir late-phase image showed significantly increased amyloid burden in the global cortex index and all six brain regions of eight AD and LV patients when compared with the other six PPA patients and eight HCs. There was no apparent uptake of amyloid tracer in both six PPA patients and eight HCs.

Conclusions: Dual-phase ^{18}F-florbetapir images of six PPA (SD and PNFA) patients showed hypoperfusion in the frontotemporal cortex, and little global amyloid uptake, which may be a distinct image pattern for differentiation among HC, AD, and PPA patients.

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Introduction

Management of dementia syndrome such as frontotemporal lobar degeneration and Alzheimer's disease (AD) depends on an early and accurate diagnosis so that an accurate prognosis and an appropriate treatment plan can be provided.[1] The diagnosis is based on clinical assessments and by the characteristic topographical distribution of brain atrophy and immunohistological pathology.[2–4] However, differentiation is sometimes difficult clinically, especially in an early stage and in presenile patients, because of the overlap of clinical symptoms and neuropsychological profiles.[2–5] Despite advances in molecular and genetic diagnostic tools, and other supportive tests, the diagnosis occasionally cannot be confirmed until autopsy.[1]

Primary progressive aphasia (PPA) is a clinical syndrome, which includes semantic dementia (SD), progressive nonfluent aphasia (PNFA), and logopenic variants (LVs), and is characterized by a progressive decline in components of the language system. Recent studies have confirmed the three clinical PPA variants according to genetic and pathological analysis.[5,6] Imaging studies have shown that elevated Pittsburgh compound B (PIB) uptake was more frequently found in AD and LV of PPA, but is uncommon in PNFA and SD.[7,8] In addition to amyloid pathology, differences between AD and PPA and the brain perfusion/metabolism features may also help in the differential diagnosis. Meta-analyses have shown reductions in metabolism and perfusion in the temporoparietal network related to episodic memory processing for AD patients, whereas reductions in the frontomedian network were associated with behavior and personality changes for PPA patients.[7–9]

Currently, ^{18}F-florbetapir (AV-45/Amyvid) is recognized as an effective amyloid-beta (Aβ)-specific radiotracer for use in positron emission tomography (PET) imaging.[10–13] Brain ^{18}F-florbetapir PET imaging assessments performed *in vivo* have been correlated with the extent of Aβ deposition found in the brain at autopsy in patients with AD, thereby validating this agent for possible use as a reliable marker for early diagnosis of AD.[10] Our previous work showed that early phase ^{18}F-florbetapir PET imaging of 1–6 minutes is potentially useful in providing complementary perfusion-like information, which makes it feasible to study in conjunction with amyloid pathology to help disease diagnosis.[14] This study was designed to compare the dual-phase ^{18}F-florbetapir in patients with PPA and AD, and healthy controls (HCs) to assess the utility of dual-phase ^{18}F-florbetapir PET for the differential diagnosis of dementia syndromes.

Methods

From August 2011 to July 2013, 22 patients including six consecutive patients with PPA (4 with SD and 2 with PNFA), eight age- and sex-matched patients with AD, and eight HCs were enrolled in this study. In addition, one LV of PPA patient was also included in the study for comparison. All patients were recruited from Chang Gung Memorial Hospital (CGMH), Taiwan. The main inclusion criteria for patients with PPA were as described by Gorno-Tempini et al[5] and others.[6–8,15,16] The diagnosis of SD is based on a fluent dysphasia with impairment of semantic verbal memory (severe difficulty in understanding the meaning of words and naming) and associative agnosia. The diagnosis of PNFA is based on expressive aphasia with word-finding phonemic paraphasia or even total mutism. All patients initially presented with language difficulty and without initial psychiatric or prominent episodic memory or behavioral disturbance. Eligible patients with probable AD who were age- and sex-matched (age range, 59–74 years) were diagnosed based on the National Institute of Neurological and Communicative Disorders and Stroke–Alzheimer's Disease and Related Disorders Association (NINCDS–ADRDA) criteria for diagnosis of probable AD.[17] Age- and sex-matched individuals (HC group) were required to show no signs of cognitive impairment, and could be either the spouse of an AD patient, a hospital volunteer, or an individual from the surrounding community. All participants received a detailed neurological examination and blood tests to rule out other systemic diseases, and underwent brain computed tomography (CT) and/or magnetic resonance imaging (MRI) to rule out the presence of other brain lesions.

The study was approved by the Governmental Department of Health in Taiwan and the Institutional Review

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